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IMPULSE FORMATION. BY THOMAS LEWIS.

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PAROXYSMAL TACHYCARDIA, THE RESULT OF ECTOPIC IMPULSE FORMATION.

By THOMAS LEWIS.

(*University College, London.*)

IN previous communications to this *Journal*, two cases of the auricular form of paroxysmal tachycardia and, in addition, certain experimental observations relating to paroxysms of rapid heart action in general have been recorded. In the following pages an account of a further example of clinical auricular paroxysms will be found with full details of the case in which the phenomenon has been observed.

The patient, who forms the basis of the remarks, originally attended the out-patient clinic of Dr. James Mackenzie. It is with pleasure that I acknowledge his generosity in placing not only the case but his own observations at my disposal.

HISTORY, ETC.

H. M. B., aged 41, a carpenter by trade and a married man, first attended the out-patient department at Welbeck Street in June, 1909, complaining of a general failure of health and attacks of giddiness and faintness. Ever since he has been under close observation.

The family history presents no point of interest. The father died at the age of 49, and the mother at 63, of ailments not definitely known. The patient has seven brothers alive and, so far as he knows, in good health. One brother was shot in South Africa. There were no sisters. Of seven children, six are alive and healthy; one died in infancy.

His *previous health* has been good, with the exception of an illness six years ago. He states that at that time he was laid up for six weeks, with influenza and right-sided pleurisy. In habit he has been neither temperate nor the reverse. Tobacco has been used sparingly. His work has been heavy and his hours long.

Present illness.—For a month previous to his admission as an out-patient he suffered from failing health. Especially, he experienced attacks of giddiness and faintness, accompanied by a frequent sense of exhaustion. He was likewise distressed by frequent cold sweats with goose skin sensation, and a curious beating, which fluctuated, at the top of the chest. A slight dry cough, shortness of breath in walking even on level ground, and sleeplessness at night complete the list of the more prominent symptoms. He has had a certain amount of feverishness at nights.

Condition. 13-7-09. The patient is a frail poorly nourished man, with sallow complexion and clammy skin. The arteries present a slight grade of arteriosclerosis. The apex beat is ill-defined, and there is no pulsation in the epigastrium. On this account records from the chest

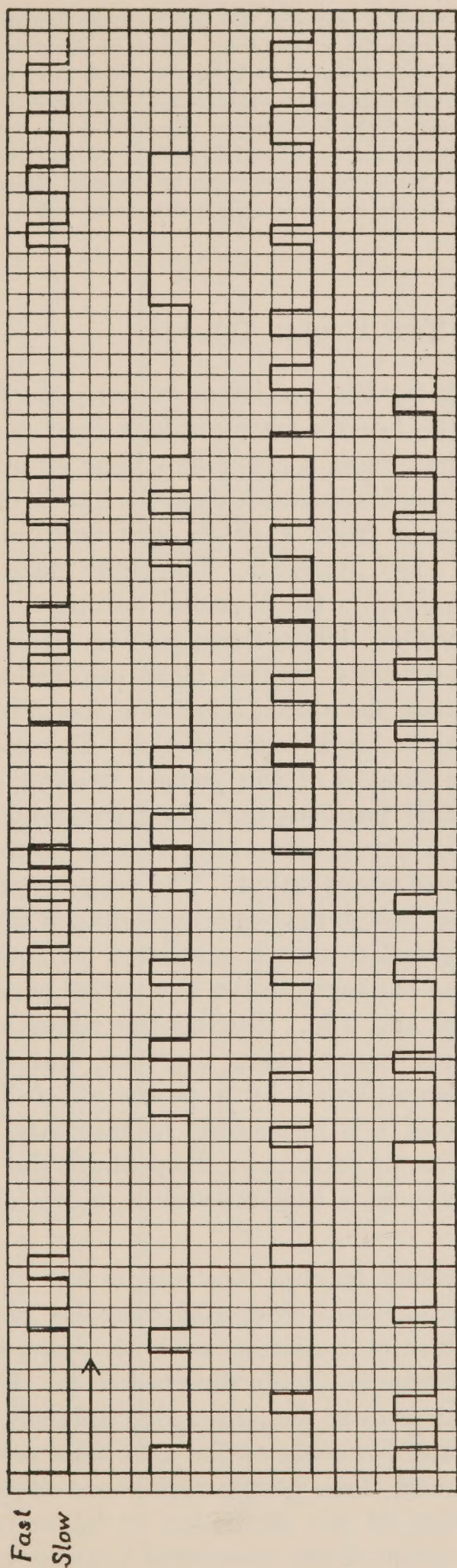


Fig. 1. The chart shows, in diagrammatic fashion, the relative duration of tachycardial and slow pulse rate. It has been constructed from a single curve, and covers 1 hour and 49 minutes. During this time 54 paroxysms occurred. The distance between adjacent ordinates is equivalent to 5 sec.. The diagram reads continuously from left to right and from above downwards in 4 lines, and the record is unbroken, except where short stops or obscure tracing are indicated by broken lines.

wall are not obtainable. The percussion limits of the heart are as follows. The left border of dulness is $4\frac{1}{2}$ and the right 1 inch from the middle line. These limits show no definite variation with the onset or offset of the brief paroxysms of tachycardia which form the chief feature of the case. The heart sounds are clear, and there are no murmurs. The movements of the chest with respiration are fair; there is, perhaps, slight deficiency at the right apex. Over this apex also the percussion note is a little but definitely impaired, and both in front and behind numerous superficial fixed crackles are audible. The breath sounds are unaltered. There are no signs of old standing basal pleurisy. The liver shows no enlargement even while the heart is beating rapidly.

THE RADIAL CURVES.

The case is remarkable for the persistent recurrence of short attacks of tachycardia. An idea of the heart's action is best conveyed by means of a diagram (Fig. 1). The chart covers a space of 1 hour 49 minutes. The four lines of which it consists read continuously. It has been constructed to scale by measurements taken from a single and continuous curve. The ordinates are separated by 5 sec. intervals. The short vertical lines represent the onset or offset of a paroxysm; the abscissæ joining the summits of such lines indicate rapid heart action, while those joining their bases correspond to the slow periods. The diagram gives a clear impression of the relative duration of fast and slow rates, the actual lengths of the paroxysmal and slow periods, and the variations in each respectively. The chart, as a whole, may be regarded as a fair sample of the picture usually presented by the patient, though it occasionally happens that the paroxysms are less frequent or of longer duration. Thus, on occasion only two or three paroxysms have been noted during an hour of observation. The longest paroxysm recorded lasted about seven minutes, but paroxysms exceeding a minute in duration are rare. The shortest recorded attack consisted of four rapid beats in the radial curve. The rate of the tachycardia has varied between 133 and 184. It usually lies between 160 and 180. During the times when the pulse is slow it is rarely regular for many beats together, but is interrupted by extrasystoles which are accompanied by a pause not fully compensatory. Therefore, on most occasions it is impossible to calculate the fundamental sinus rhythm with any degree of certainty. Sometimes the pulse is perfectly regular over lengths of curve of a half minute or more, and then the rate lies between 76 and 88.

A short strip of the radial tracing is given in Fig. 2. It shows three paroxysms breaking into the usual slow and irregular rhythm of the slow periods. The patient is usually conscious of both onset and offset of the tachycardia, when his attention is closely directed to it. While lying supine he was requested to raise his arm at the onset and to lower it at the offset of each attack. The raising and lowering of the arm were marked by hand on the curve as it was taken, and these rough signals are shown on the accompanying curve. Recognition of the alteration of heart rate was attained by the patient by his perception of the associated beating in his chest.

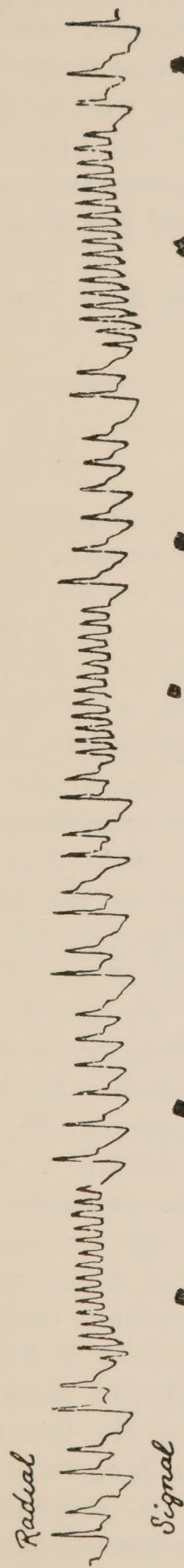


Fig. 2. Radial pulse curve, taken at a comparatively slow rate. It shows three short paroxysms of tachycardia. The slow periods are irregular. The signal records the patient's appreciation of the onset and offset of each separate attack. The delay is approximately 2 sec. at both onset and offset.

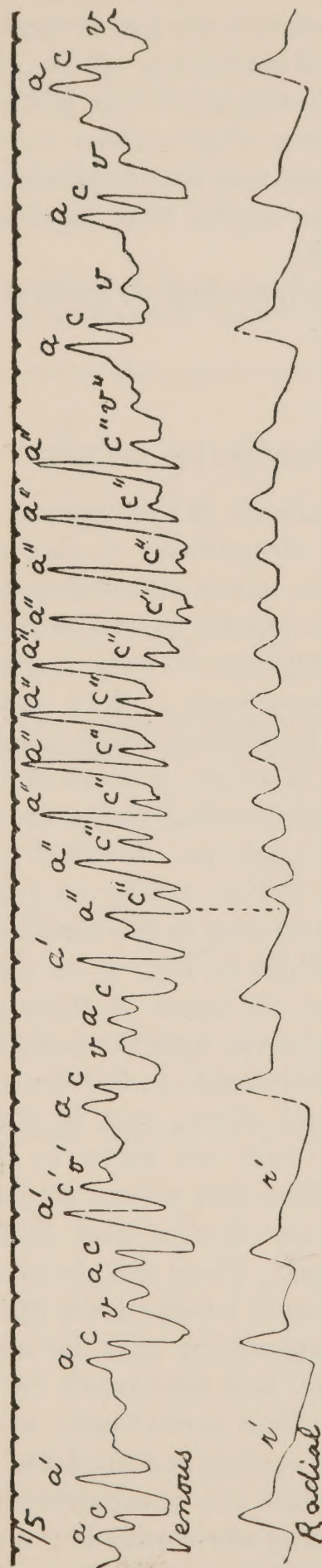


Fig. 3. Faster curve taken on the same day. Venous and radial curves are shown. The slow period at the commencement is interrupted by auricular extrasystoles (a'). The paroxysm is started in the auricle, as evidenced by the waves a'' . There is a slight rise of B. P. subsequent to the onset of the attack.

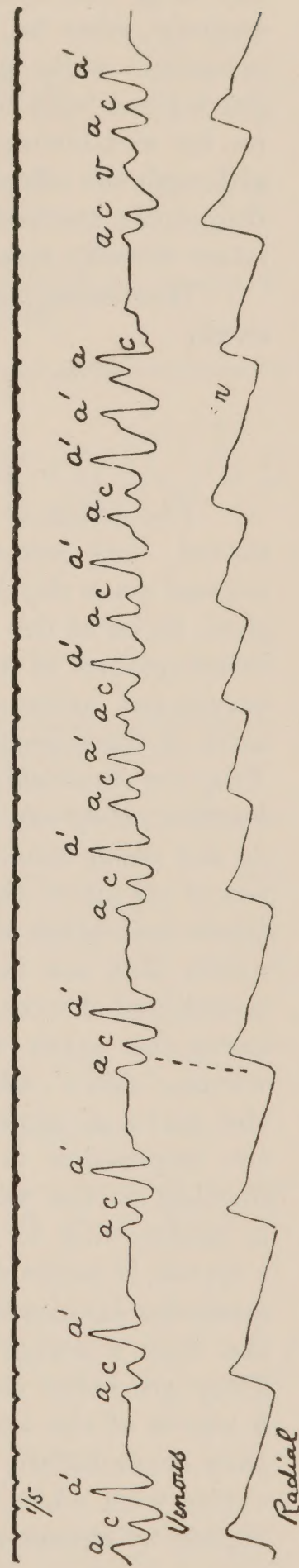


Fig. 4. Four auricular extrasystoles, probably awakening no ventricular response, but interfering with the sinus rhythm, are shown to the left. They are succeeded by interpolated auricular extrasystoles, in which neither sinus nor ventricle are affected.

The attacks are most frequent when the patient has been at work, and after exertion of any kind. They tend to subside, but never disappear entirely, after he has been recumbent for several hours or longer. On one occasion, while under the observation of Dr. Mackenzie, and while the paroxysms were relatively infrequent, it was noticed that they were brought on by swallowing. Numerous observations were made at the time, and although the effect of a swallow was not invariable, yet the movement was so frequently succeeded by a paroxysm that no doubt remained but that the latter directly resulted from it.

Treatment, apart from improving his general health, has been without avail.

POLYGRAPH RECORDS.

The action of the heart between the paroxysms is, as has been previously stated, very irregular. The irregularity is due to the interruption of the normal sinus rhythm by beats, which simultaneous radial and venous curves show to be of the nature of auricular extrasystoles. The frequency of these interruptions of the sinus rhythm is very variable; there may be only occasional extrasystolic contractions; they may alternate with one or more of the normal beats, or may occur frequently and in haphazard fashion. *They are invariably auricular in origin*, and are almost always very numerous. Further, they are isolated and (leaving the paroxysms out of consideration) do not occur successively or in groups. Studied in the radial curve they are found to affect the pressure in the artery to a small extent or not at all (some indication of them is to be seen in the radial curves of Figs. 3, 5, 6 and 8, where they are marked r' , while in Fig. 4, as also in several of the above mentioned figures, they leave no trace of their presence). In the venous curve the extra wave a' is always well marked, and it is often followed by waves c' and v' , which are attributable to the ventricular response. At times the wave a' appears to stand alone, and in these instances (cp. Fig. 4) the impression is conveyed that we have to do with a failure of conduction to the ventricle. That the waves a' , in Fig. 4, for example, are in reality due to auricular contraction, and that they are not anomalous v waves, is easily demonstrated. They are identical with the a' waves of the auricular extrasystoles, and occur earlier after the preceding c wave than do the true v waves*. The comparison may be made in Fig. 4, or in Fig. 6. They are taller than v waves, and also reach to a greater height than the a waves of the sinus beats. This is obviously attributable to the fact that they occur before ventricular systole is complete. Lastly, the lengths of the cycles with which they interfere are compatible with those of cycles interrupted by extrasystoles reaching the ventricle; and they are unaccompanied

* Attention is specially directed in this connection to the relation of the fall of the wave, a' or v as the case may be, to the preceding c wave.

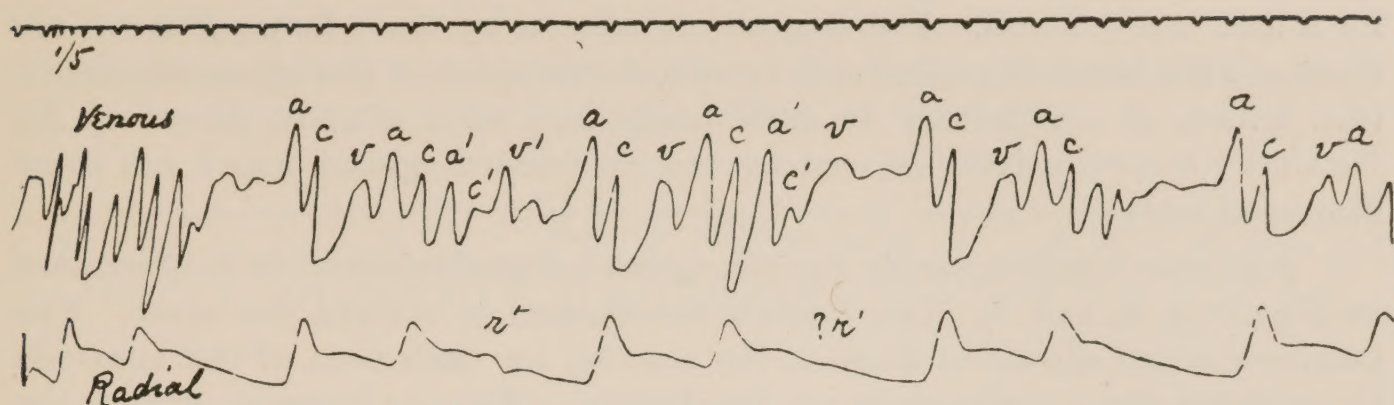


Fig. 5. Venous and radial curves during a slow period. Auricular extrasystoles are shown. The stops may be used in checking measurements in this and other figures.

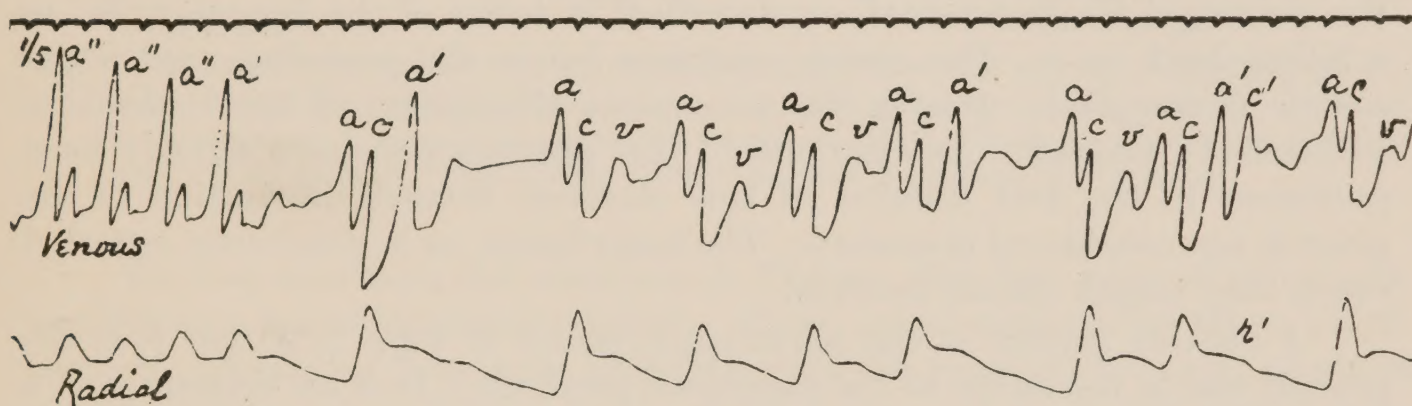


Fig. 6. The end of a paroxysm, showing the termination in a long pause, which is succeeded by a gradually accelerating sinus rhythm, itself interrupted by auricular extrasystoles.

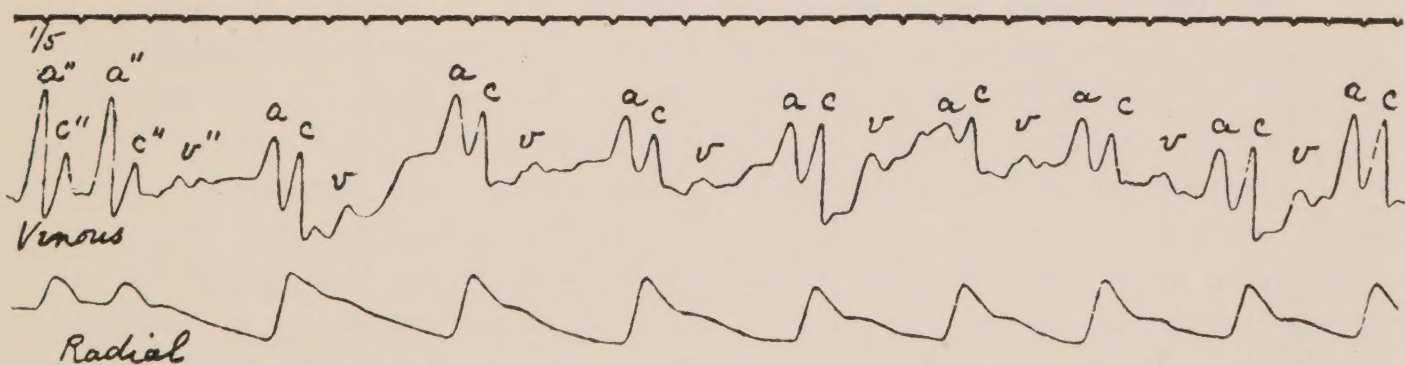


Fig. 7. The same as Fig. 6, except that during the slow period no extrasystoles occur.

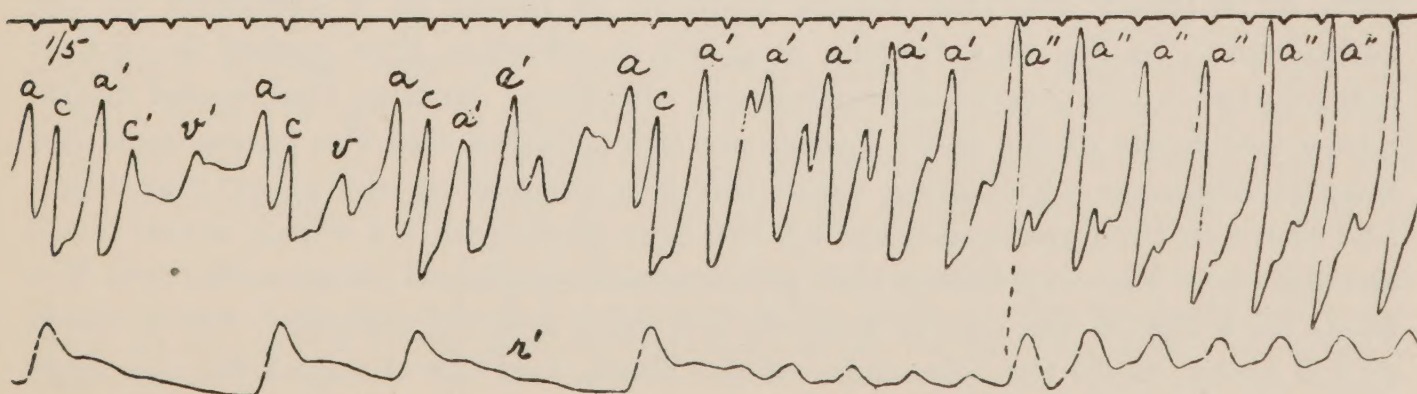


Fig. 8. The onset of a paroxysm. The slow period to the left is irregular as a result of auricular extrasystoles. Tachycardia starts in the auricle, towards the centre of the figure; it consists of a series of extrasystoles (a') each awakening an abortive ventricular response. The curve changes abruptly to the true paroxysm (a'').

by a first heart sound. The suggestion, offered by these facts, that we are dealing with blocked auricular extrasystoles in some of the examples shown (the failure of conduction of such beats has been already described by Hewlett²) is conclusively proved by the electrocardiograms which are to be discussed later.

The heart's action during the paroxysms is equally clear. It is illustrated in Figs. 2, 3, 8, and 9. The heart's mechanism is always the same. The paroxysms are shown to start in the auricle, for each beat of the ventricle is preceded by a contraction of the former. The *a-c* interval during the paroxysms is about $\frac{1}{3}$ sec.,* that is to say, it is of about the same length as during the slow periods. The excessive height of the *a* waves is assigned to the shortened Vs-As interval, or, described in terms of the venous pulse, to *a* falling back on *v*. The auricle contracts before the preceding ventricular systole is complete. During the paroxysms alternation of the ventricle is occasionally present. As examples of this phenomenon have already been published in the first number of this *Journal*, further illustration of the point is not considered necessary. The lesser beats, as in previously reported cases, may vanish almost entirely.

A notable feature of the paroxysms, shown in Figs. 2, 3, and 8, is the general rise in the curve as the paroxysm proceeds. It is an indication of a slight increase of blood pressure consequent upon the more rapid heart action.

The transitional portions of the curves are of special interest. The paroxysms commence in a variety of ways. The auricular extrasystoles are always frequent directly before the onset. Fig. 3 shows a comparatively simple form of onset. The slow rhythm is disturbed by *effective* auricular extrasystoles (or extrasystoles which cause ventricular responses). Two of such beats are shown in the figure. Following these beats are two normal responses, then an auricular extrasystole, and from this point onwards regular auricular waves appear (*a''*) each of which awakens a ventricular response. In some instances two or more extrasystoles may directly precede the paroxysm, and in such cases a long pause in the radial curve, with or without abortive waves upon it, is found as a precursor of the paroxysm. In Fig. 8 a tracing is given in which a slow period is at first interrupted by extrasystoles which are effective; during the second radial pause two beats of the same nature are interpolated and the pause ends in a normal response. Following upon this is a succession of five extrasystoles, and these in turn are succeeded by the true paroxysm (of which seven beats are shown). The expression true paroxysm is employed because the first five beats lie lower in the curve, they do not raise arterial pressure, which tends to fall away, as do the remainder of the beats, and the change from one type

* There is a slight variation in the intervals in many of the curves, more especially the electrocardiographic, which tends to show that the normal As-Vs distance is greater with the abnormal than with the sinus beats (cp. Fig. 17), but it is not always well marked.

to the other is marked by an abrupt rise in the curve. Further reasons for this differentiation will be stated later. The same phenomenon is visible in the opening beats of the paroxysm shown in Fig. 9.

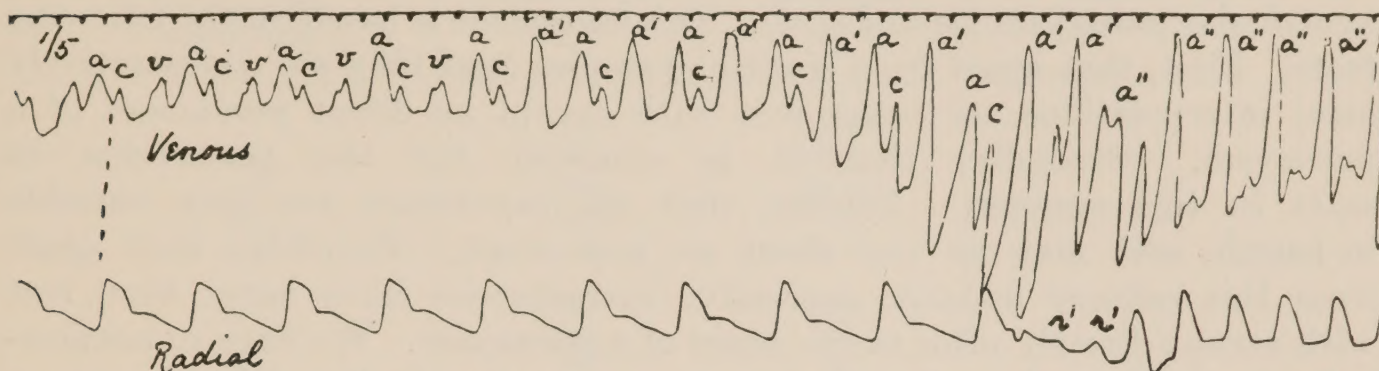


Fig. 9. Onset of a paroxysm. The rhythm and sequence is at first normal. Interpolated auricular extrasystoles appear suddenly in the curve. None of these reach the ventricle. Between the interpolation stage and the paroxysm proper (of which 5 beats are shown) two abortive radial curves are seen. They are the result of successive auricular extrasystoles.

We may now turn our attention to Fig. 4. The first beats of this curve are blocked auricular extrasystoles*. These extrasystoles, like those which awaken ventricular responses, interfere with the sinus rhythm and produce the diastolic pause indicated in the radial pulse. At times the disturbance of the sinus rhythm is absent and on such occasions *interpolated auricular extrasystoles* occur. Fig. 4 illustrates the condition very clearly, and shows the sudden passage of one mechanism to the other. Moreover, they are interpolations of a very curious nature. For they affect neither sinus nor ventricle. Every interpolated beat is blocked†. The disturbance is strictly limited to the auricle, and the regular impulses transmitted across this chamber to the ventricle are in no way interfered with‡. At the end of the interpolation period the radial pulse shows an increased pause. It is probable that at this time two auricular extrasystoles occur, and that the last beat of the pair alone yields a ventricular response, though the response is doubtful. Fig. 9 confirms the explanation adopted in the case of Fig. 4. The tracing commences with the normal sinus rhythm; each ventricular contraction is associated with *a*, *c*, and *v* waves. Later, in the curve auricular extrasystoles suddenly appear, and they affect neither sinus nor ventricular rhythms. Apart from the mode of its onset, the curve, up to this point, is in every way similar to Fig. 4. Towards the end of the curve also the extra beats are seen in succession, and they give rise to two small radial waves, *r'* and *r''*.

* The evidence for the blocking of these particular beats is, perhaps, inconclusive: the statement is based on the fact that blocked extrasystoles are known to occur frequently in this case (from an examination of the electrocardiographic curves), and upon the absence of c' and v' waves in the curve.

† This is clearly the case, for otherwise the a and a' waves would be equally prominent.

‡ More recently records have been obtained from another patient, in which interpolated auricular extrasystoles were present, but in which each extra beat was transmitted to the ventricle.

The curve then passes into the paroxysm proper in which the auricular beats are evenly spaced and give rise to regular responses. From these curves, and from a large number of others in my possession, it is recognised that Fig. 4 represents the preliminary stage which often precedes the appearance of the ventricular paroxysm (as in Fig. 9). This statement is based on the following facts. First, that apart from similar examples (and they are comparatively rare) interpolations are never met with except as direct precursors of a paroxysm. Secondly, that it is common for the paroxysms to start in this manner. Thirdly, that the paroxysms are very variable in length, and may be cut short at any stage. Fourthly, that apart from this solitary instance successive extrasystoles have never been met with except directly prior to the onset of a paroxysm. We have to acknowledge in the first place that the paroxysms are foreshadowed by the occurrence of an auricular tachycardia, consisting of alternate responses to sinus and alternate interpolations, and that this premonitory tachycardia is of a very similar rate to that of the paroxysm itself (though it is rarely identical with it). At the same time we have evidence that the paroxysms and the interpolation periods are essentially different, though closely bound up together. This evidence as at present advanced is as follows. Periods of interpolation are always separated from paroxysm by an interval, in which one, two or more of the insignificant r' waves appear in the radial pulse. Secondly, beats of the paroxysm itself are never blocked.

The termination of the paroxysms is of equal interest. It is illustrated in Figs. 3, 6, and 7. The paroxysms invariably end in a prolonged pause. The length of this pause is variable within certain limits, and is occasionally interrupted by the occurrence of a smaller beat of the type seen at the commencement of the paroxysms. As a rule such interruptions are absent. Careful measurements were made in several strips of curve in which paroxysms were frequent, for the purpose of ascertaining the relation between the pause following the paroxysm and the pause following extrasystolic contractions during the slow periods. On account of the variation in presphygmic interval known to accompany extrasystoles, the measurements were made in the venous curves from a' or a'' waves to a waves. The results are embodied in the table. In the first column the length of the pause following an auricular extrasystole is given. The extrasystole was chosen from the centre of a slow period, and may, therefore, be said to succeed one paroxysm and precede the next. In the fourth column the pause following the succeeding paroxysm is given. It will be seen that the pauses vary slightly and in approximately the same degree, and that the figures given in the two columns are in close agreement*.

The average for the paroxysmal pauses exceeds that for the extrasystolic by $\frac{0.07}{5}$ sec., a practically negligible quantity. In a previously reported case of paroxysmal tachycardia, considerable stress was laid upon the occurrence

* The variation in individual cases is scarcely greater than the error in measurement.

TABLE OF PAUSES.

Length of pause in $\frac{1}{5}$ sec., following AEs, between the paroxysms.	Rate of paroxysm per minute.	Length of paroxysm in sec..	Length in $\frac{1}{5}$ sec. of pause at end of paroxysm.
—	169	16.2	4.5
4.4	161	23.5	4.5
4.5	166	16.6	4.3
4.6	157	3.2	4.3
5.2	165	5.2	4.5
4.0	182	19.4	4.4
4.4	180	4.6	4.3
4.4	181	5.9	4.5
4.6	163	6.5	4.4
4.7	169	6.9	4.6
4.5	170	8.7	5.0
4.4	167	8.0	4.0
4.1	165	4.8	4.1
4.4	167	4.2	4.6
4.2	168	6.1	4.9
4.1	169	5.5	4.3
4.1	170	5.4	4.1
4.5	170	6.1	5.1
4.3	165	4.8	4.5
4.4	166	6.4	4.4
4.4	167	6.0	4.6
4.5	167	4.3	4.7
4.4	165	4.2	4.6
Average 4.41	—	—	4.48

of these pauses, but the wide variation then observed in them would not allow of their co-relation with any other measurement. The reason of the difference in the two patients is probably to be found in the fact that we are at present dealing with short paroxysms, while in the earlier case the tachycardia always persisted for a long while. The cause of the pause is now more obvious. It may be regarded as identical in extrasystole and paroxysm, and may be interpreted as the time taken for the last impulse to reach the sinus, plus the time taken for the formation of a fresh sinus impulse (Cushny and Matthews¹). The length of the post-paroxysmal pause would be readily accounted for in this way were it not that an additional phenomenon is present. Fig. 7 shows the termination of a paroxysm in uninterrupted normal rhythm. The end of the paroxysm is succeeded by a decreased pulse rate, followed by a return to the normal. A slow sinus rate as a sequence to the paroxysm is invariable, but it is fugitive. Similar retardation of the pulse at the termination of a paroxysm has been noticed as of constant occurrence in another patient; it was absent in the case previously reported, and also in the instance of the paroxysms produced experimentally.

The phenomenon reminds us of the preliminary slow rate of a newly awakened idioventricular rhythm, and of the gradual development of a faster and more constant rate. The explanation is insufficient in that it fails to account for the absence of prolongation of the post-paroxysmal pause. It might be attributed to a reflex increase of vagal tone, and the absence of retardation in the experimental instance would harmonise, for the vagi were divided. The facts do not warrant anything but the most tentative conclusion; emphasis is laid upon the slowing, because it stands in contrast to the acceleration which often follows the single extrasystole. Its presence at the commencement of the slow period fully explains the variation in the lengths of the beats of the *interrupted* rhythm which usually succeeds the paroxysms (an example of which is shown in Fig. 6). It served as an additional reason for the measurement of extrasystolic pauses lying in the centre of the slow periods only.

ELECTROCARDIOGRAPHIC CURVES.*

We may now conclude the description of the investigation of this case by referring to the electrocardiographic curves. So far, and in the main, we have been able to elucidate the following facts. We are dealing with a tachycardia of auricular origin, and the abnormal mechanism during both paroxysmal and slow periods is attributable to ectopic beats arising in auricular tissue. We have also very suggestive evidence that we have to do with ectopic beats of several kinds. This suspicion at once becomes a conclusion with the galvanometer curves before us. There are at least three varieties of ectopic beats.

The interpretation of the accompanying curves, depends largely upon the view adopted as to the nature of the electric curve yielded by an auricular contraction starting in an abnormal position in the auricle. In my first paper upon paroxysmal tachycardia the possibility that an abnormal auricular curve would result was fully recognised, and in the second paper the proof of the proposition was furnished by the example of a retrograde contraction. Fig. 10 shows simultaneous venous and electrocardiographic curves, and allows of a very exact analysis. The first two beats, which are shown, are of normal sinus origin. But there is a notable difference between the T waves of the two ventricular systoles. The second lifts more abruptly, is shorter, and is followed by a prolonged pause. The venous curve shows that an auricular extrasystole has occurred, and it has been ineffective. The interpretation is completely borne out by Fig. 12, where the auricular extrasystole though effective produces precisely the same deformation of the wave T. To proceed with Fig. 10, the next two beats are normal, the following

* The electrocardiographic curves were taken at University College Medical School, and the expenses involved in their production have been defrayed by a grant from the British Medical Association.

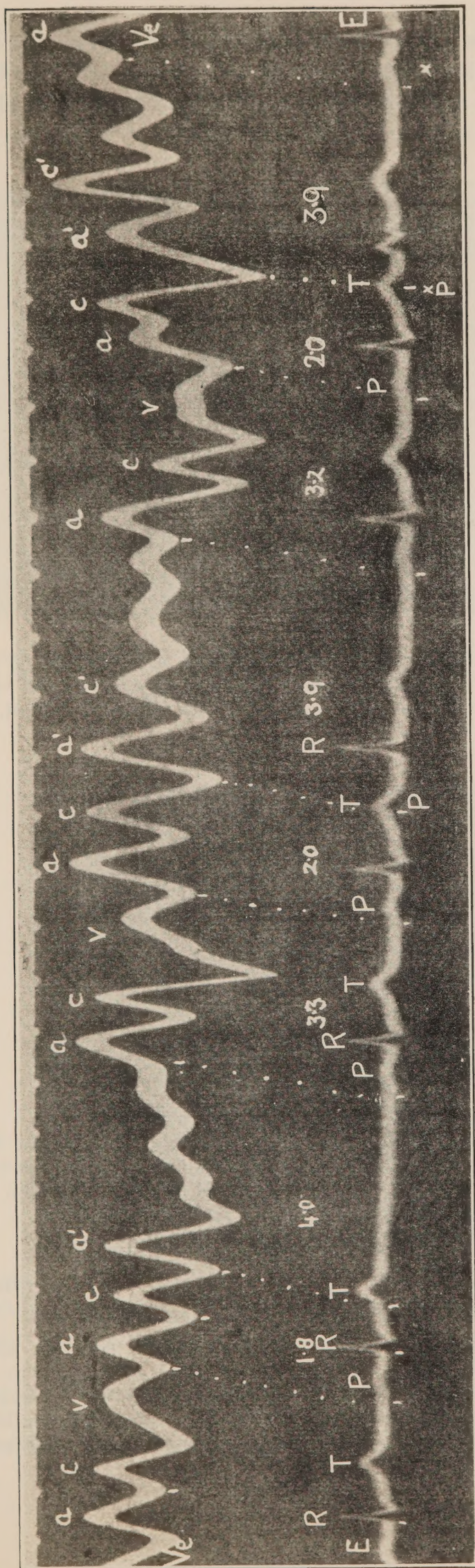


Fig. 10. Simultaneous venous (Ve) and electrocardiographic (E) curves. As in all figures the time (top curve) is in $\frac{1}{5}$ sec.. From left to right, two normal beats, a blocked auricular extrasystole, a long pause, two normal beats, an auricular extrasystole (Type I), a pause, two normal beats, an auricular extrasystole (Type II), a pause and a commencing normal beat, are shown. Each auricular extrasystole deforms the preceding T wave. The space figures are in terms of fifths of seconds.

one is premature. It is an auricular extrasystole (Type I), and it likewise deforms the preceding T wave*. But the alteration of this T wave by the auricular contraction is different from that of the T wave of the second beat,

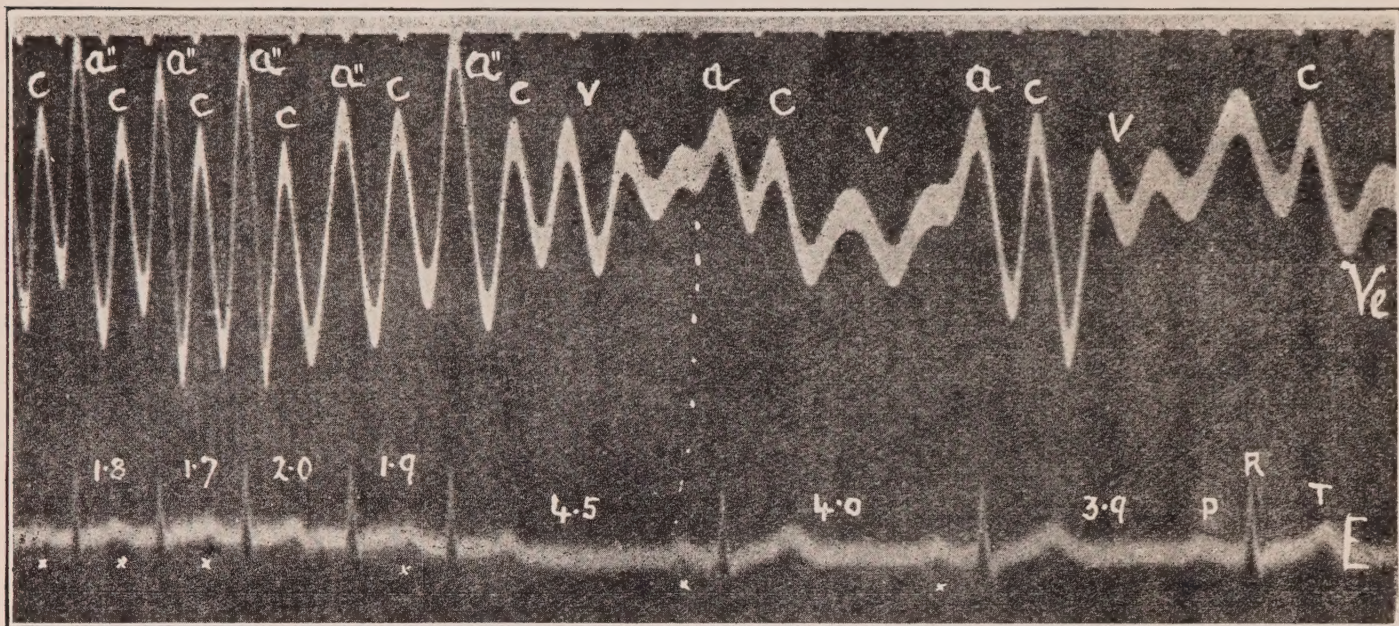


Fig. 11. The end of a paroxysm. Acceleration is well shown in the slow period. As in Fig. 10, and in succeeding figures, small white crosses are placed beneath downward notches in the curve, which represent auricular contraction.

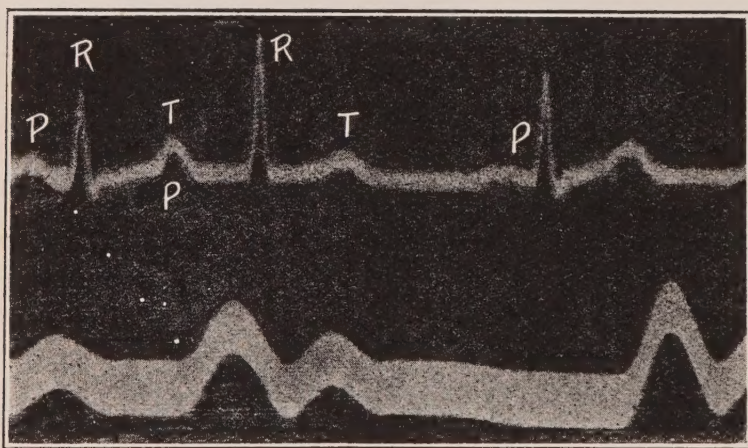


Fig. 12. Electrocardiogram and radial curve. A normal beat, in which the auricular notch is well-marked, is succeeded by an auricular extrasystole of Type I. It causes the deformity of the first T wave. The extrasystolic R is large. Prolongation of the As-Vs interval is distinct in the case of the extrasystole.

and this is due, as is the efficiency of the auricular contraction in creating a ventricular response, to the later occurrence of the extra beat. The next two beats of the curve are normal. They are succeeded by the third auricular

* The peak R of the extrasystole is taller than that of the normal beats, yet the pulse beat accompanying such beats is often absent (Fig. 12). The same disproportion has been met with clinically in heart alternation, the large peak R may correspond to the small pulse beat.

extrasystole (Type II) to be found in the curve. It is known to be auricular by an examination of the accompanying venous curve, and from the fact that it deforms the T wave which lies to the left of it*. We have proof, therefore, of auricular extrasystoles of two types, and may now turn

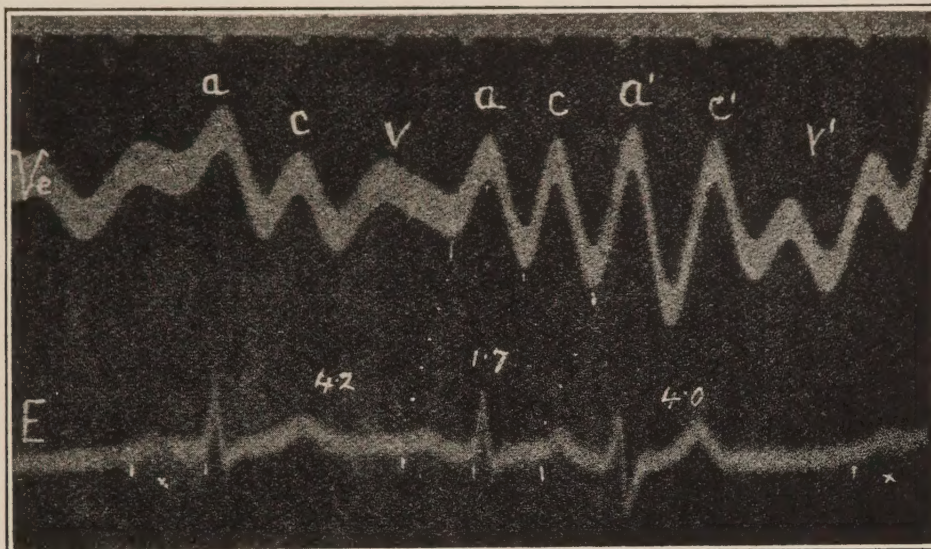


Fig. 13. Venous and electrocardiographic curves. Two normal beats are succeeded by an auricular extrasystole of Type III.

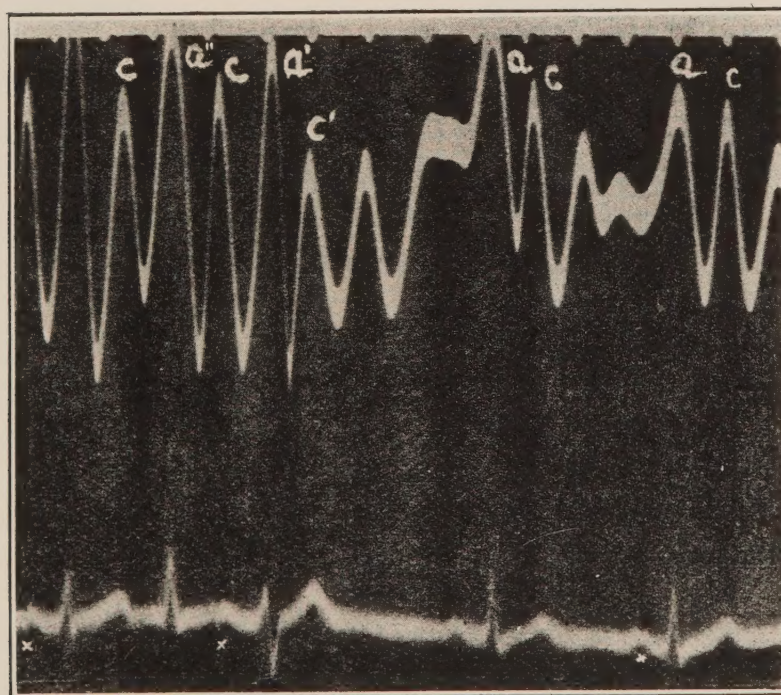


Fig. 14. The end of a paroxysm. The paroxysm terminates in an auricular extrasystole of Type III.

* The dotted line drawn to T is not quite correctly placed. Its upper end should terminate more to the right. The upstroke, to the bottom of which it is drawn, is in reality the upstroke of *v*. The correction may be checked from the peaks of the venous waves.

to Fig. 13. In this curve an example of the third type (Type III) is shown. Precisely similar arguments to those already employed demonstrate it as an auricular extrasystole. The same form of beat occurs in Fig. 14, where it terminates the paroxysm*. Attention is now drawn to a character of the curves, which, though unexplained, is of peculiar service. We have seen that the deformation of the wave T by extrasystolic auricular beats consists of a slight notching in the positive or downward direction. This notching is far more distinct in some curves than in others, and is clearer in the originals than in the reproductions. Moreover, it is plainly seen in many of the *normal P curves* (cp. Figs. 10, 11, 14, 15, 16, and 17). It serves to identify the auricular contraction, but while it occurs in the centre of the normal auricular curve P, it appears to be the sole representative of the abnormal auricular contractions, and is situated at the starting point of these beats†. In the accompanying curves the notch has been marked, where clearly visible in the originals, by a small white cross.

The paroxysms themselves, portions of which are shown in Fig. 11 (first part), Fig. 14 (first part), and Fig. 16 (last part), consist of beats bearing a close resemblance to the extrasystole of Type I (and shown in Figs. 10 and 12). The paroxysmal type is shown most clearly in the last four beats of Fig. 16. The apparent difference between beats of this nature and the extrasystolic type shown in Fig. 12, consists in the deformation of the associated T waves by the deep notching resulting from the auricular contractions. The same general features are visible in Figs. 11 and 14. Now the type of extrasystole referred to (Type I) is the rarest of all during the slow periods. The commonest type is that shown at the end of Fig. 13 (Type II), and the third type with the deep S depression is almost as common. As it may be said that the paroxysm is constituted by beats‡ similar to or identical with the rare type of extrasystolic beat, emphasis may be laid on a caution formerly expressed. This was to the effect that the conclusion, that beats of a paroxysm and those interrupting the slow periods are identical, is not warranted in the absence of galvanometric evidence.

In concluding a description of the facts presented by this case, several other points must be noted. Fig. 15 is an example of an alternation of

* Termination in this manner is quite exceptional.

† Explanations parallel to those which have been offered for the split P wave in mitral stenosis naturally suggest themselves, but they would lead at present to deductions too vague to be of value.

The absence of any indication of auricular contraction other than the dip (which is obviously a special feature in this particular case) throws a new light upon the case reported in this *Journal*, p. 51. It is no longer possible to attribute the wave marked P in Fig. 5a to auricular contraction alone. There is every possibility that it may represent a T or deformed T wave. This reservation affects the remarks made in the first paragraph of p. 70.

‡ The paroxysmal beats are always of this type. It is remarkable that in three cases of auricular paroxysmal tachycardia, the ventricular portions of the paroxysmal electrocardiographic curves have always been of the normal type (Type I) and never of the abnormal types (Types II and III). They differ from the normal beats in the corresponding patients by the increased prominence of R, and this despite the faster heart rate prevailing.

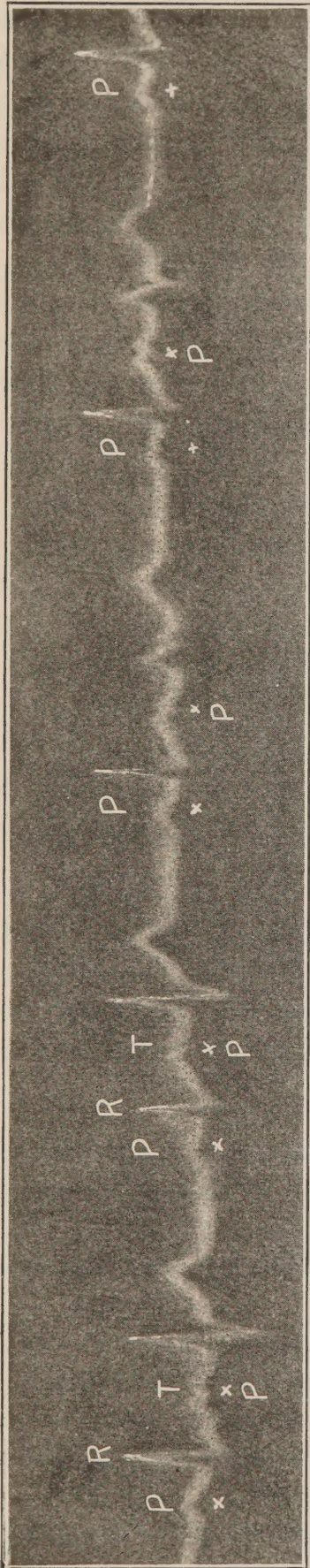


Fig. 15. Electrocardiographic curve showing alternate normal and extrasystolic contractions. The latter are all of auricular origin. The auricular notches are very clear. The "bigeminy" abruptly alters. It contains beats of Type III at the beginning, and of Type II at the end.

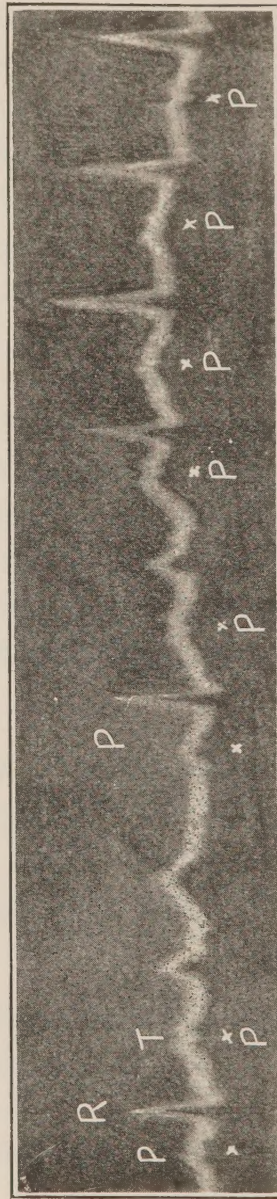


Fig. 16. From a curve continuous with Fig. 15. The onset of a paroxysm (last 4 beats) is shown. The second auricular extrasystole (of Type II) is shown directly preceding the paroxysm.

normal beats and auricular extrasystolic contractions; the remarkable feature of this curve is the sudden change in the type of the ectopic beats. While a continuous "bigeminy" is present, it is of one type to the left, and of another and distinct type to the right*. The curve directly preceded a paroxysm, of which the commencement is seen in Fig. 16. The time tracing unfortunately failed in these curves, but the spacing of the beats can be gauged from the rate of the paroxysm shown at the end of Fig. 16. The second extrasystole of Fig. 16 commands especial attention. The beats adjacent to it are separated by an interval approximately equal to that between alternate beats of the paroxysm itself. It might be regarded as an interpolation, but it differs from those shown in Figs. 4 and 9 in two respects. In the first place it is effective, and in the second place the P' wave belonging to it is separated from the preceding P wave by a shorter interval than from the succeeding P wave. The beat belongs to those discussed as precursors of the paroxysm of Fig. 9. The presence of this beat at the commencement of the paroxysm helps to justify the previous conclusion that those insignificant beats at the commencement of tachycardia which contrast so markedly in the radial curve with the beats of the paroxysm proper are of a different nature to the paroxysmal beats†. The differentiation is of importance, for, if it is accepted‡, we may conclude that only those beats which appear in the fully developed form in the radial curve are paroxysmal. When a paroxysm of four rapid beats appears in the radial curve, even if it is preceded by tachycardia in the auricle, we have to assume that they are the offspring of four auricular ectopic beats, and that these four alone belong to the ectopic rhythm proper.

The interpretation of the curves involves two assumptions; first, that the auricular tachycardia displayed by Fig. 4 is not an ectopic rhythm, but consists of interpolations. This is evident from the curves which precede such periods, and from the strict separation of such periods from the paroxysms proper.

The second assumption is that the paroxysm itself does not consist of interpolations. It is perfectly obvious that it is not composed of

* Several different combinations of this change in the type of the bigeminy have been noted in this patient.

† Considerable stress is laid on the radial differentiation; experience shows it to be of great value. The appearance of successive extrasystoles directly before the onset of a paroxysm, and at this point only, has recently been observed in another patient, and the electrocardiographic evidence is in this instance conclusive. A suggestion is offered that the preliminary successive extrasystole may constitute the exciting cause of the paroxysm itself.

‡ Later observations upon the case here reported have conclusively shown the nature of the beats referred to. Several electrocardiographic curves demonstrating the onset of a paroxysm in successive beats of Type II have been obtained.

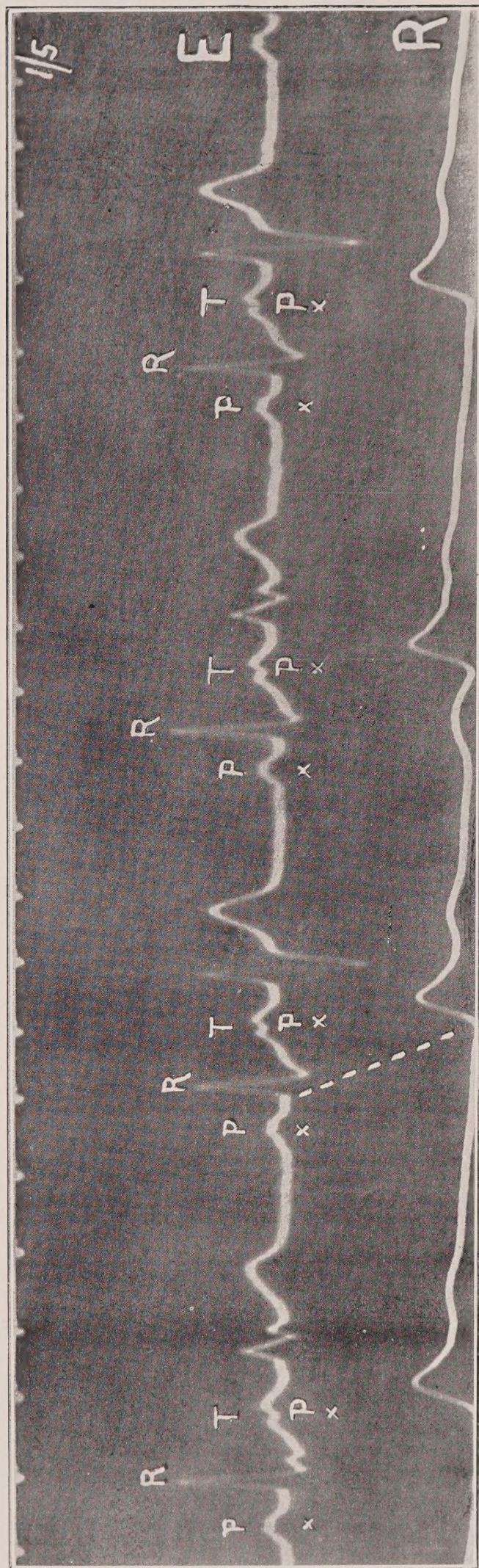


Fig. 17. $\times \frac{1}{5}$ linear. Electrocardiographic and radial curves. A "bigeminy," consisting of normal beats and auricular extrasystoles, is shown. The abnormal beats are of Types II and III; they are arranged alternately between the normal beats.

interpolations of the two commoner types, but it is more difficult to exclude a series of interpolations of the type shown in Fig. 12. Nevertheless we are justified in excluding this possibility also. When this type of beat occurs singly it does not affect the arterial pressure to any appreciable extent (Fig. 12). Interpolations when found in other parts of the tracings are never effective in producing ventricular response. Further, the beats of the paroxysm are never blocked, although the paroxysmal rate is subject to considerable variation. Again, the beats of the paroxysm are quite regularly placed, and the radial curves do not exhibit alterations in excursion which would be expected if alternate beats were produced by distinct mechanisms. Finally, if this view of interpolation is adopted the beats of the paroxysm which have to be assumed as of normal sinus origin may be easily ascertained, and, as a result, it is found that the last beat of the paroxysm has at times to be assumed as a normal beat, and at times as an extrasystolic contraction. If such were actually the case the uniformity of the post-paroxysmal pause would not be expected.

Fig. 17 is an example of the alternation of two types of extrasystole in a bigeminal curve. Several combinations of the three types have been met with, and they produce similar pictures.

In this figure the notches, representative of auricular contraction, are particularly well marked. But they are more clearly shown in the case of the extrasystolic contractions. On the other hand, in Figs. 15 and 16 the depressions are equally conspicuous in both. Fig. 17 was taken at a later date.

GENERAL DISCUSSION.

The present instance of paroxysmal tachycardia is remarkable for one feature at least. It forms a useful connecting link between those instances in which paroxysms of an hour or a day are the rule, and cases in which short runs of ectopic beats are alone found. In a previous paper I have expressed the conviction that there is no essential difference in the nature of the individual beats of the forms of paroxysmal tachycardia considered, and the beats hitherto spoken of as extrasystolic contractions, and have brought forward experimental evidence in favour of this view.

In the present case we have an example of paroxysmal tachycardia in which the ectopic rhythm, upon which it depends, may last for four beats only or for several minutes. The fact that at one time but a few beats appear,

and that at another a long continued succession of beats is found, cannot, therefore, be held as an essential or distinguishing factor. The chain of instances is now complete from the single extrasystole to the groups of several successive extrasystoles, to instances of paroxysms of short duration, and eventually to those which are prolonged for days or even weeks.

The second feature of importance in the present case is the level at which the ectopic rhythm arises. We may certainly place the focus of origin in the auricular tissue, and in consideration of the abnormal character of the electric curve may conclude that the auricle has contracted in an abnormal direction. This is tantamount to a conclusion that the starting point of auricular systole lies at some distance from the normal centre of impulse formation, a conclusion which receives support from the length of the pause following the individual paroxysms. If we accept the upper regions of the auricle as the normal centre of impulse formation, we must, as a consequence, place the origin of the present paroxysms in the middle or lower level of the auricle. The last-mentioned region is improbable, but the reasons for this opinion cannot be entered upon at the present time; they are based upon the detailed examination of another instance of ectopic paroxysmal tachycardia which will be fully described at a later date. Suffice it to say that in the new instance the auricular curve in the electrocardiograms is completely inverted, and that there is strong presumptive evidence that the seat of origin is in the lowest level of the auricle. The view previously expressed that ectopic rhythms may arise at any level of the heart is thus confirmed.*

The appearance of three separate types of ventricular electrocardiogram, each as a result of an auricular extrasystole, remains to be discussed. It is a fact for which a complete explanation fails at the present time, and involves questions which can only be solved by appeal to experiment. It has been customary to assume (1) that all beats of the ventricle of supraventricular origin are conveyed to that chamber along a single path, and that they enter the ventricular musculature by the same doorway. It has also been supposed (2) that all systoles of the ventricle which arise at a fixed point in the musculature will, *cæteris paribus*, yield the same and a distinctive type of electrocardiogram. Therefore, it is further assumed that all beats of supraventricular origin will yield the same type of electrocardiogram. The evidence of this case is directly opposed to the last conclusion, and there seems to be no alternative but to seek for some flaw in the propositions upon

* The conclusion, based in part on an undescribed case, is necessitated, for the case in question will be utilised subsequently in the discussion of an entirely different problem.

which this conclusion is based. Both the original propositions (1 and 2) are supported by considerable evidence, evidence which is well known and which it would be out of place to discuss at the present time. Kraus and Nicolai have stated that auricular extrasystoles started experimentally in right and left auricle are accompanied by ventricular electrocardiograms of different forms. Their observation, with which the present clinical facts are consistent, is equally at variance with our present views of the cardiac mechanism, or with our present interpretation of electrocardiographic curves.

The several types of beat described in this paper cannot be explained as the result of varying time relations. They cannot be identified except by electrocardiographic means. A suggestion which can be offered in explanation of them is that they may arise from separate foci in the auricular walls; the location of the exact sites of origin of the impulses is beyond the limits of this communication.* In regard to the questions which their presence raises the writer inclines to the opinion that the explanation must be sought in a revision of our present ideas of the possible mode (or modes) of conduction from auricle to ventricle.

SUMMARY AND CONCLUSIONS.

1. A case of paroxysmal tachycardia, due to ectopic impulse formation, is described, in which it is believed that the source of impulses lies at a point near the physiological centre of the main mass of auricular tissue. The case is exceptional in that the paroxysms are short and of extremely frequent occurrence. The case forms a connecting link between other instances in which either single ectopic beats or ectopic rhythms of long duration interfere with the normal mechanism of the heart. The case supports the views already adopted that there is no essential distinction between extrasystolic and paroxysmal beats, and that paroxysmal tachycardia which is due to ectopic impulse formation may arise at any level of the heart.

2. Auricular extrasystoles met with clinically give rise to ventricular contractions of at least three distinct forms, as evidenced by the electrocardiographic curves which they yield.

* The question of accurate location is the more important as very similar, if not identical, types have been observed, singly or together, in other cases. In other words, the present case is by no means phenomenal in this respect.

3. A clinical instance of blocked auricular extrasystoles (Hewlett) and of interpolated auricular extrasystoles is described.

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